Dear all,

We have sent you an open letter and a request for a meeting. Below you will find a scientific information pack that hopefully will give you a deeper understanding of Borrelia (spirochete bacteria), Bartonella (intracellular bacteria), and Babesia (intraerythrocytic parasites), three chronic pathogens found in both humans and animals, oftentimes called the 3B as they often co-occur in patients.

Lyme Suisse is an association of patients and caregivers. Our association is supported by more than thirty municipalities in French-speaking Switzerland. Our website, www.lyme.ch, which has already been visited more than 2,000 times in less than a year, summarizes and popularizes the knowledge from our "One Health" database, which contains over 470 recent articles about the 3B in humans and animals. Lyme Suisse has set up a telephone hotline, which was overwhelmed in just two months, and a self-assessment questionnaire for chronic patients, which has already been completed by almost 200 patients. Our association offers occasional discussion groups and manages two social networks (Facebook and Instagram).

Lyme Suisse is in contact with countless patients who have been unable to find medical help. We have enabled them to obtain a diagnosis of one or more of the 3B thanks to specialized American doctors and laboratories, now recognized by the College of American Pathology and the Food and Drug Administration. These doctors and laboratories are members of the International Lyme and Associated Diseases Society (ILADS.org), an organization that for 30 years has been caring for chronically ill patients who have been neglected by hospital infectious disease departments: the latter are too busy dealing with “hot” acute infections (with fever) and neglect "cold" chronic infectious symptoms (without fever). It is important to note that in less than 20 years, hundreds of "Lyme" associations have sprung up around the world, representing millions of members who believe that current healthcare systems have proven incapable of understanding their symptoms, finding the causes of their disorders, and offering targeted and effective treatments.

European Lyme disease experts have focused on tick-borne encephalitis virus and Borrelia, in its acute dermatological or joint manifestations, and have stubbornly believed that the latter can be easily eradicated with a few weeks of single-agent antibiotic therapy. They never considered that **Borrelia spreads deep into connective tissues and protects itself with biofilms that are impervious to antibiotics**. They also failed to take into account **co-infections, particularly Babesia and Bartonella**, which are also persistent. These two organisms target **red blood cells** and/or **endothelial cells**, settle in the blood of patients, and induce numerous systemic, diffuse symptoms as well as overwhelming and debilitating asthenia, which is completely misunderstood by the medical profession. These pathogens, which are notoriously underdiagnosed, colonize both vessels and tissues. They contribute to blood cytokine profiles of "cold" inflammation without CRP (elevated TNF-α, IL-1β, **IL-6**, IL-12, IL-17, IFN-γ), which are nonetheless symptomatic and deleterious. The 3Bs are responsible for immense suffering and place a significant burden on our society and healthcare system. This suffering has been given too many names: **chronic Lyme disease, fibromyalgia, chronic autoimmune and inflammatory diseases, chronic fatigue syndrome/myalgic encephalomyelitis, psychiatric disorders such as ASD, ADHD, GAD, OCD, and schizophrenia**.

Our goal is to share major scientific advances and ensure that they can no longer be ignored in Switzerland. **We are writing to call for an urgent update of diagnostic methods and treatment protocols for chronic Borrelia, Babesia, and Bartonella infections (the "3Bs").**

→ turn to discover Borrelia

1. **Borrelia: underestimated prevalence and systemic impacts**

According to a systematic review and meta-analysis published in 2022, the overall seroprevalence of Borrelia is estimated at 14.5%, with high rates in Central Europe (20.7%) and Western Europe (13.5%) (PMID: 35697507). In Switzerland, chronic Borrelia infections are undoubtedly underdiagnosed (PMID: 36113496) because screening tests first of all do not target Borrelia from the TBRF subgroup. Then, Swiss serological tests are indirect, i.e., based on the host's immune response. Unfortunately, this response is greatly modified by Borrelia itself (the more a mammal is colonized, the less its immune system produces immunoglobulin G - PMID: 37782044). Borrelia colonizes most connective tissues and establishes itself there by deep infiltration and the creation of biofilms, causing **very low-grade local inflammation over a very long period of time.** It expresses its own enzymes, but also activates metalloproteinases 3 and 9 (PMID: 11119537), which allows it to degrade its host's extracellular matrix (PMID: 10583909).

This process of slow paracrine degradation allows Borrelia to feed and inexorably alters the fibrous structure, shape, and therefore function of the colonized organs and nerves. A striking example of this could well be low back pain, which costs the healthcare system and disability insurance system in Switzerland an incredible amount of money. How many of these highly disabling and recurrent cases of low back pain are caused by Borrelia (PMID: 33690837)? In recent weeks, Borrelia has been implicated in vertebral hypermobility[1] in mice, which could well explain many cases of low back pain, sciatica, and cervical disorders in chronically infected humans. What proportion of these radicular neurological disorders could be controlled by intermittent intramuscular anti-Borrelia treatments (PMID: 40267215)? **The dermis, adventitia, capillary connective tissue, Purkinje fiber sheaths, pericardium, perineurium, meninges, joint capsules, ligaments, tendons, and fascia are colonized by this spirochete**, which needs neither iron nor oxygen to breathe, but uses, in the deep tissues it colonizes, far from immunity and oxygen, a whole reduced physiology based on manganese and the use of amino acids and fatty acids produced by the relentless degradation of the connective and nervous tissue surrounding it. Why not consider developing loratadine treatments that prevent Borrelia from using this critical manganese (PMID: 25709405, 41230968) as soon as a patient is known to be colonized. Loratadine has also been shown to make antibiotic therapies much more effective (PMID: 38075920).

The presence of Borrelia is now correlated with numerous **cardiac disorders** (PMID: 40267215), **invasive breast cancer** (PMID: 38451280), and **chronic lymphocytic leukemia** (PMID: 37871679). It should also be noted that Borrelia has been found to be colocalized with **amyloid markers** in the brain tissue of **Alzheimer's disease** (PMID: 34897095) and that ApoLipoE, the amyloid protein typical of Alzheimer's disease, appears to be an immune protein deposited in response to the presence of a contiguous threat. This threat could well be discreet Borrelia biofilms containing, with age and degradation of the upper jaw, more and more oropharyngeal bacteria whose surface antigens would stimulate immunity and the expression of ApoLipoE (PMID: 40559130)... Numerous studies in the USA are currently moving in this direction.

It becomes clear that Borrelia, its biofilms that can be colonized by an even more inflammatory pathobiome, as well as the catabolic and autoimmunogenic paracrine environment it induces by promoting the presentation of self-antigens by dendritic cells (PMID: 33043033), could very likely be involved in many chronic and auto-immune diseases that are overwhelming our hospitals. Early diagnosis and lifelong monitoring of the Borrelia load in the body (if eradication proves impossible) could prevent costly disabilities and avoid many chronic diseases (PMID: 37405694 & 38578312). Faced with complement inactivation by Borrelia (PMID: 32482556) and with the slow invasion of Borrelia into the tissues, the adaptive immune system stubbornly attempts to overcome a pathogen whose surface proteins vary in order to evade immunity (PMID: 39792948). Countless successive

[1] https://doi.org/10.1093/jimmun/vkaf283.1711

waves of different antibodies are produced against Borrelia throughout a lifetime: increasingly less specific and increasingly cross-reactive. The switch from IgM to IgG which characterizes a successful adaptive immunity is inexorably prevented by an ever-increasing bacterial load (PMID: 37782044). The immune system can only produce IgM confined to the intravascular space, and the tissues are no longer perfused with 'anti-Borrelia' IgG. Fibrous tissues accumulate even more Borrelia. The natural immune system tries to compensate (PMID: 38578312). Its continuous activation leads to a decrease in tolerance of self-antigens (PMID: 21920710): **everything points to the onset of an autoimmune disease.**

It becomes clear that thinking one can eradicate 100% of Borrelia from an individual with a short course of single-agent antibiotic therapy is almost magical thinking. Many of these organisms are deeply infiltrated and protected, inaccessible to antibiotic molecules (PMID: 35467428). This is why patients' migratory joint pain and asthenia reappear, often **~3 months after the end of their treatment with a single antibiotic. This is the time it takes for the slow but persistent surviving spirochetes to recolonize their host's connective tissue**.

Two recent publications by Dr. Richard Horowitz, a world expert in the clinical management of chronic Lyme disease, demonstrate that **dapsone in combination with other intracellular antibiotics (cyclines, macrolides, rifamycins) eradicates Borrelia in 9 weeks in patients who have been colonized and ill for more than 20 years** (Double Dose Dapsone Combination Therapy for 8 weeks followed by High Dose Dapsone Combination Therapy for 4 to 6 days = DDDCT + HDDCT). These publications suggest that Bartonella can also be eradicated by increasing the number of HDDCT sessions, extending them from 4 to 6 days, or adding pyrazinamide. Unfortunately, these case series found that some patients relapsed quickly after HDDCT (~3 days): all were found to be carriers of Babesia, whose rapid reproductive cycle leads to a rapid resurgence of symptoms.

→ turn to discover Babesia

2. **Babesia: a flagrant underdiagnosis and the explanation for "chronic Lyme disease" with collapse, disability, medical wandering, refusals of disability benefits, and often suicide**

Babesia, an intraerythrocytic parasite transmitted by ticks, **is simply not diagnosed in Switzerland**, despite its frequent co-occurrence with Borrelia in ticks (PMID: 38061320). Medical personnel in Switzerland are simply not trained to recognize or treat this infection. Laboratories are also not equipped to detect the various species of Babesia: they offer antigen tests for Babesia microti or PCR tests that are not sensitive enough.

Sequestrating Babesia species such as **Babesia odocoilei** are now implicated in severe chronic Lyme disease (PMID: 38992682). Colonized red blood cells display Babesia proteins on their surface: these proteins enable red blood cells to adhere to each other and to endothelial cells. These clusters of "sticky red blood cells" reduce blood flow in the microcapillaries and promote fibrin deposition (PMID: 10417157). **Babesia hiding in these "clusters" avoid destruction by the spleen.** The multiple Babesia colonies, located throughout various microcapillaries in the body, contribute to the establishment of a **chronic infection that is inaccessible to the spleen's immune system**. These sequestering Babesia create transient but systemic circulatory symptoms that are shifting but highly debilitating in many organs (brain, lungs, kidneys). Transient localized hypoperfusion, functional hypoxia, tachycardia, anxiety, panic attacks due to "shortness of breath" linked to slow blood flow in the pulmonary capillaries and mild pulmonary edema (PMID: 10386441 & 38133320), mitochondrial dysfunction, extreme fatigue, and a brain fog of indescribable intensity. These symptoms are strangely reminiscent of certain autoimmune diseases that are now treated long-term with antimalarial drugs such as hydroxychloroquine. A coincidence? Perhaps not. It turns out that **Babesia, a cousin of malaria, must be treated with antimalarial drugs** (PMID: 37764145 & PMID: 38792737), often over the long term. Several studies have shown that this parasite can be resistant to standard treatments (atovaquone + azithromycin) and that it is often necessary to add primaquine or tafenoquine to eradicate Babesia and prevent chronically infected patients from relapsing (PMID: 31319461 & 38814096).

According to many ILADS researchers and physicians, most cases of "chronic Lyme disease" will turn out to be undiagnosed babesiosis. This would explain what many chronic Lyme patients have been saying for so long: long-term treatment with macrolides, cyclines, and rifamycins revives them, but a rapid relapse (~3 days) occurs as soon as they stop taking them. These intracellular antibiotics certainly inhibit the functions of the apicoplast, a "bacterial" organelle essential for Babesia replication. Long-term treatment with antibiotics hence lowers parasitemia; the number of "nests" decreases, circulation resumes, IL-6-mediated inflammation subsides, and physical and psychological symptoms improve. However, dormant Babesia parasites always remain, ready to reproduce rapidly as soon as antibiotic therapy is stopped.

Sequestrating babesiosis leads to brain fog that is unimaginable to the average person. All those with chronic Lyme disease who **have lost their concentration, memory, job, or spouse** can attest to this. The vast majority receive no diagnosis, treatment, medical recognition, or disability benefits. These abandoned patients wander in total despair, some even resorting to suicide. The thousands of daily posts by "chronic Lyme sufferers" on social media reveal nothing less than an **epidemic that has been ignored by the medical profession**. Babesia symptoms are often worsened by undiagnosed Bartonella.

→ turn to discover Bartonella

3. **Bartonella**: a silent epidemic with **unexpected immunopsychiatric impacts**

There are more than **2 million cats in Switzerland. And nearly half a million dogs**. Many people are inevitably exposed to bites and scratches from these animals, as well as bites from their fleas, which often carry Bartonella spp (B. henselea, B. vinsonii, B. elizabethae, B. clarridgeiae, B. koehlerae). Bartonella is also transmitted by ticks (PMID: 18598628).

American specialists such as Prof. Breitschwerdt no longer hesitate to describe Bartonella as **"an epidemic hidden right under our noses"** (PMID: 39369199 & 30911227). Some even believe that Bartonella and the generalized anxiety it causes are probably **responsible for a substantial proportion of psychiatric hospitalizations in both adults and children** (PMID: 37941969 & 38911703).

Bartonella, after an acute feverish phase often mistakenly attributed to influenza or mononucleosis, then goes into **"crypto-infection" mode**. It no longer produces immunostimulatory surface lipopolysaccharides, **but settles intracellularly within various cell lines**: endothelial cells and erythrocytes, of course, but also macrophages, monocytes/mononuclear phagocytes, microglia, dendritic cells, CD34+ progenitor cells, myeloid angiogenic cells, cerebral vascular pericytes, mesenchymal stromal cells, dental pulp stem cells, hepatocytes, basal keratinocytes, and fetal brain cells (PMID: 22232371). **It multiplies very slowly (one division per 48 hours)**, immunomodulates its host by interfering with the presentation functions of dendritic cells and the phagocytosis functions of macrophages (PMID: 34975788), **and creates extracellular colonies protected by biofilms in many tissues.** It is therefore surprising that in Switzerland, the only cases of bartonellosis diagnosed are those following a a cat scratch with a swollen lymph node or an endocarditis. The latter is detected by PCR on replaced heart valves (PMID: 30886729). These 2 manifestations are only the tip of the iceberg.

In 2025, it seems reasonable to think that Bartonella, by invading endothelial cells and pericytes (PMID: 23184416), **disrupts the sorting function of the blood-brain and blood-cerebrospinal fluid barriers,** allowing numerous systemic inflammatory cytokines and many small antibodies normally confined to the bloodstream to pass into the cerebral parenchyma and cerebrospinal fluid. The latter interfere with synaptic functions. Since October 2025, Borrelia has also been found to disrupt the endothelial cell-pericyte interface (PMID: 41125572). These two pathogens could therefore be working together to alter what the human brain is chronically exposed to.

The mammalian brain is composed of neurons, but also of 12% immune cells. It seems almost inevitable that a **systemic mast cell activation syndrome** due to the presence of one or other of the 3Bs will cause the release of excessive amounts of histamine. This 'hyperhistaminergic' state also opens the blood-brain barrier. When microglial cells—the mast cells of the brain—are exposed to histamine and systemic cytokines elicited by the 3B, they can also activate and degranulate histamine, flooding the brain with histamine. This **overload** has direct effects: hypervigilance, insomnia, anxiety, panic disorder, PTSD (PMID: 37201895). Since November 2025, the 3Bs have even been correlated with bipolar disorder in children[2] .

Immunopsychiatry, an emerging science, posits that most psychiatric conditions result from functional changes caused by the direct effect of inflammatory cytokines but also **the interference of antineuronal antibodies with various neurotransmitter receptors**[3] . Our impulses govern our behavior from childhood onwards. Our adaptability and ability to integrate into human society requires a form of control over our emotions and impulses. In social mammals, many dopaminergic neurons in the basal ganglia are involved in inhibiting impulsive behaviors and rewarding inhibitions that have enabled group cohesion. The sum of these inhibitions is an integral part of our personality, our controlled, adaptable, and socially integrated "self." Inevitably, a brain flooded with anti-dopamine receptor antibodies, whose dopamine receptors in the striatum are blocked, will be

[2] https://doi.org/10.3389/frcha.2025.1685016
[3] https://www.moleculera.com/neuropsychiatric-symptoms

incapable not only of feeling reward, but also of respecting codes of conduct and providing the series of strategic inhibitions necessary for socio-professional success (PMID: 31791436). It is even likely that such a brain, impaired in its dopaminergic functions, will be diagnosed with a psychiatric disorder or exhibit compulsive, even aggressive (Bartonella rages) or criminal behavior (PMID: 38200989).

Many psychiatric patients have **Bartonella streaks** (Bartonella-Associated Cutaneous Lesions or **BACL**; PMID: 33291688). These skin lesions are rarely detected by hospital staff or institutions caring for vulnerable people; **BACL are still too often mistaken for stretch marks**. Patients infected with Bartonella receive heavy psychotropic treatments that mask the psychiatric and physical symptoms that are indicative of Bartonella (retrosternal or intratibial pain for example) and only conceal the very real distress and very biological symptoms. Eradicating Bartonella with two very long-term intracellular antibiotics like a macrolide and a rifamycin restores the function of the blood-brain barrier, improve neuropsychiatric symptoms, and prevents overwhelming anxiety, psychological suffering, ostracism, and the many cardiovascular deaths now observed in people with schizophrenia (PMID: 36756905). Many have never been diagnosed with Bartonella. **Today, many regulatory disorders, addictions, compulsive and aggressive tendencies (cutting in teenagers) are attributed to Bartonella (PMID: 38200989)**. Most shelters for drug addicts (PMID: 8944742) or homeless people (PMID: 35749315) in Switzerland should clinically screen their users for BACL and have the means to confirm this infection by FISH. If these highly vulnerable populations are unable to manage their daily lives, it may well be because they are too busy drowning their anxiety, psychiatric symptoms, and immune-induced cognitive impairment in alcohol or drugs.

Finally, it should be noted that Bartonella and Babesia target the same compartment, red blood cells, creating a more pronounced, severe, and complex clinical picture in cases of concomitant infection. This appears to be linked to significantly reduced cerebral perfusion and even more severe encephalitis of the basal ganglia, possibly due to Babesia's eukaryotic surface proteins eliciting more neurological auto-immunity. **Although invisible, a co-infection with Bartonella and Babesia is highly debilitating (PMID: 40083671)**. **We should know how to diagnose it**, and it should **quickly** qualify **for disability benefits** as long as it remains untreated.

Finally, let us add that another pathogen targets endothelial cells and leads to chronic debilitating fatigue: SARS-CoV-2. Interestingly, it too appears to be a potential co-infection of Bartonella (PMID: 38472519).

→Turn to discover the newest diagnostic methods for the 3B

SWITCHING TO DIRECT TESTS FOR A HEALTHCARE REVOLUTION

Indirect tests (serology, Western Blot) for 3B are obsolete.

At the end of 2025, North American researchers at the forefront of 3B diagnosis describe **serological tests as "laughable."** These tests have low sensitivity because chronic cold infections such **as 3B can inhibit the adaptive immune response** that is supposed to control them. Borrelia blocks the production of IgG immunoglobulins, which are the only ones capable of penetrating deep into the tissues to neutralize it, preventing the important switch from large IgM in the blood to small IgG in the tissues (PMID: 38514468).

It is urgent that Switzerland invests in **direct screening for Borrelia (including TBRF) as soon as it becomes available** in order to be able to provide patients with the primary etiology of their disorders: this will drastically reduce the number of cases of psychiatric, autoimmune, rheumatological, and chronic inflammatory diseases and lead to **substantial healthcare savings**.

Implementing this revolution will require a considerable increase in the number of infectious disease specialists and the creation of entire departments dedicated to “cold” chronic infections. But the investments will quickly reduce the size and costs of the immunology, neurology, rheumatology, and even adult and child psychiatry departments.

DIAGNOSTIC TESTS TO BE PREFERRED IN 2026

1. Borrelia: Acesdiagnostics.com/lymeseek will detect 10 antigens simultaneously with >90% sensitivity for many different species of Borrelia at various stages of the disease. Direct test available in 2026[4].

In the meantime, the use of indirect tests such as recombinant IgM and IgG protein immunoblots (average sensitivity 93%) validated by the FDA[5] , such as those from **the IgeneX** laboratory, remain a good alternative to the current Swiss ELISA + WB tests (average sensitivity estimated at 53%; no better than a coin toss). These immunoblots enable the identification of numerous Borrelia species (sensu lato & TBRF[6]).

2. Bartonella: **TLabDX's** revolutionary direct **RNA-FISH** test, which directly visualizes microbial RNA in the blood and **proves the presence and metabolic activity of these intracellular pathogens**[7]. Bartonella striae (BACL) should be considered as a direct and evident clinical proof of infection, especially in psychiatry (PMID: 33291688).

3. Babesia: **TLabDX's** revolutionary direct **RNA-FISH** test, which directly visualizes microbial RNA in the blood and **proves the presence and metabolic activity of these intracellular pathogens**[8].

[4] medscape.com/viewarticle/new-lyme-blood-test-bests-standard-diagnostics-detecting-2025a1000kmj
[5] igenex.com/fda-clearance
[6] igenex.com/igenex-immunoblot-test
[7] tlabdx.com/about-us/#molecular
[8] tlabdx.com/about-us/#molecular